Mechanical circulatory support for patients with infarct-related cardiogenic shock: a state-of-the-art review

Jacob Eifer Moller ,[1] Holger Thiele ,[2] Uwe Zeymer,[3] Alastair Proudfoot,[4] Christian Hassager[5]

[1]Department of Cardiology, Odense Universitetshospital, Odense, Denmark
[2]Department of Cardiology, Heart Center Leipzig-University Hospital, Leipzig, Germany
[3]Herzzentrum Ludwigshafen, Ludwigshafen, Germany
[4]Barts Health NHS Trust, London, UK
[5]Cardiology, University Hospital of Copenhagen, Copenhagen, Denmark

Correspondence to
Professor Jacob Eifer Moller; jacob.moeller1@rsyd.dk

Received 19 October 2024
Accepted 1 December 2024

Check for updates

© Author(s) (or their employer(s)) 2025. No commercial re-use. See rights and permissions. Published by BMJ Group.

To cite: Moller JE, Thiele H, Zeymer U, *et al*. *Heart* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/heartjnl-2024-324883

ABSTRACT

Acute myocardial infarction-related cardiogenic shock (AMI-CS) is a severe, life-threatening condition characterised by inadequate tissue perfusion due to the heart's inability to pump blood effectively. The pathophysiology of AMI-CS usually arises from the sudden loss of myocardial contractility, leading to a decrease in cardiac output and systemic hypoperfusion. In approximately 90% of AMI-CS cases, the left ventricle is the primary site of dysfunction.

Despite early recognition and the implementation of strategies such as primary percutaneous coronary intervention, the mortality rate associated with AMI-CS remains alarmingly high, reflecting significant unmet clinical needs. A major challenge lies in identifying the optimal patient population for mechanical circulatory support (MCS) devices, as these interventions are costly and can lead to serious complications.

This review provides a comprehensive overview of the pathophysiological mechanisms underlying AMI-CS, explores the current range of MCS devices available and offers an in-depth discussion on the balance of benefits and risks associated with these devices. By highlighting key evidence from recent studies, we aim to shed light on the clinical decision-making process and improve outcomes in this high-risk patient population.

Acute myocardial infarction-related cardiogenic shock (AMI-CS) is a severe, life-threatening condition characterised by inadequate tissue perfusion due to the failure of the heart to pump blood effectively. Despite advances in early revascularisation and medical therapies, AMI-CS remains a leading cause of mortality in patients with AMI, with 30-day mortality rates still ranging from 40% to 50%.[1 2] Incidence rates of AMI-CS have decreased with better prevention and treatment of AMI, but the condition still affects approximately 5–10% of patients presenting with ST-segment elevation myocardial infarction (STEMI).[1 3] However, the number of patients with non-STEMI-CS and the proportion of patients presenting with postcardiac arrest haemodynamic instability are increasing.[1 3]

PATHOPHYSIOLOGY

The pathophysiology of AMI-CS in absence of mechanical complications usually stems from the abrupt loss of myocardial contractility, leading to decreased cardiac output and systemic hypoperfusion (figure 1). The left ventricle (LV) is affected in 90% of AMI-CS cases and hypoperfusion will trigger a cascade of compensatory mechanisms.[4] To maintain perfusion, sympathetic drive is increased with release of endogenous catecholamines, which exacerbates myocardial oxygen depletion, leading to further contractile dysfunction, organ hypoperfusion and metabolic acidosis. As a result, multiple organ systems, including the kidneys, liver, gut and brain, are compromised due to tissue dysoxia. Furthermore, inflammation and endothelial dysfunction often complicate and compound the clinical picture further.[5] A significant proportion of patients with AMI-CS will experience an inappropriate vasodilation in the course of shock pathophysiology. Typically, AMI-CS will occur in a non-dilated LV in prior non-congested patients; this contrasts the patient with acute-on-chronic heart failure shock. Thus, in the patient with AMI-CS, pulmonary congestion is related to an abrupt rise in left ventricular filling pressure rather than fluid overload.

UNMET CLINICAL NEEDS

The high mortality rate in AMI-CS is clearly underscoring the significant unmet clinical needs. One challenge is optimal patient selection for mechanical circulatory support (MCS) devices. In most randomised MCS studies 40–50% of controls survive without MCS.[6 7] Thus, 50% of an AMI-CS population may primarily be exposed to device-related complications. Further, some patients will not benefit from MCS due to refractory shock. Thus, there is a need to identify the patient with a state of persistent hypoperfusion caused by left ventricular failure early in their clinical course. This can be particularly challenging where there is overlap with AMI and out-of-hospital cardiac arrest (OHCA). Many of these patients remain comatose after resuscitation needing intubation, and vasopressor is often needed to counteract the vasodilatory and cardiodepressive effects of sedation.[8] Further, no-flow and low-flow states during resuscitation will elevate blood lactate levels. Thus, admission lactate may be more reflective of low flow during resuscitation rather than a state of hypoperfusion at hospital arrival, and there is no clear relationship between lactate levels and cardiac output in comatose patients with OHCA.[9 10]

The application of risk scores to identify patients most likely to benefit from MCS is clinically challenging; current risk scores often rely on variables not available at point of care. This inherent time bias may delay decision-making around MCS, specifically prior to percutaneous coronary intervention

Heart: first published as 10.1136/heartjnl-2024-324883 on 16 January 2025. Downloaded from http://heart.bmj.com/ on February 2, 2025 at .

SHOUT
SHARE
IT

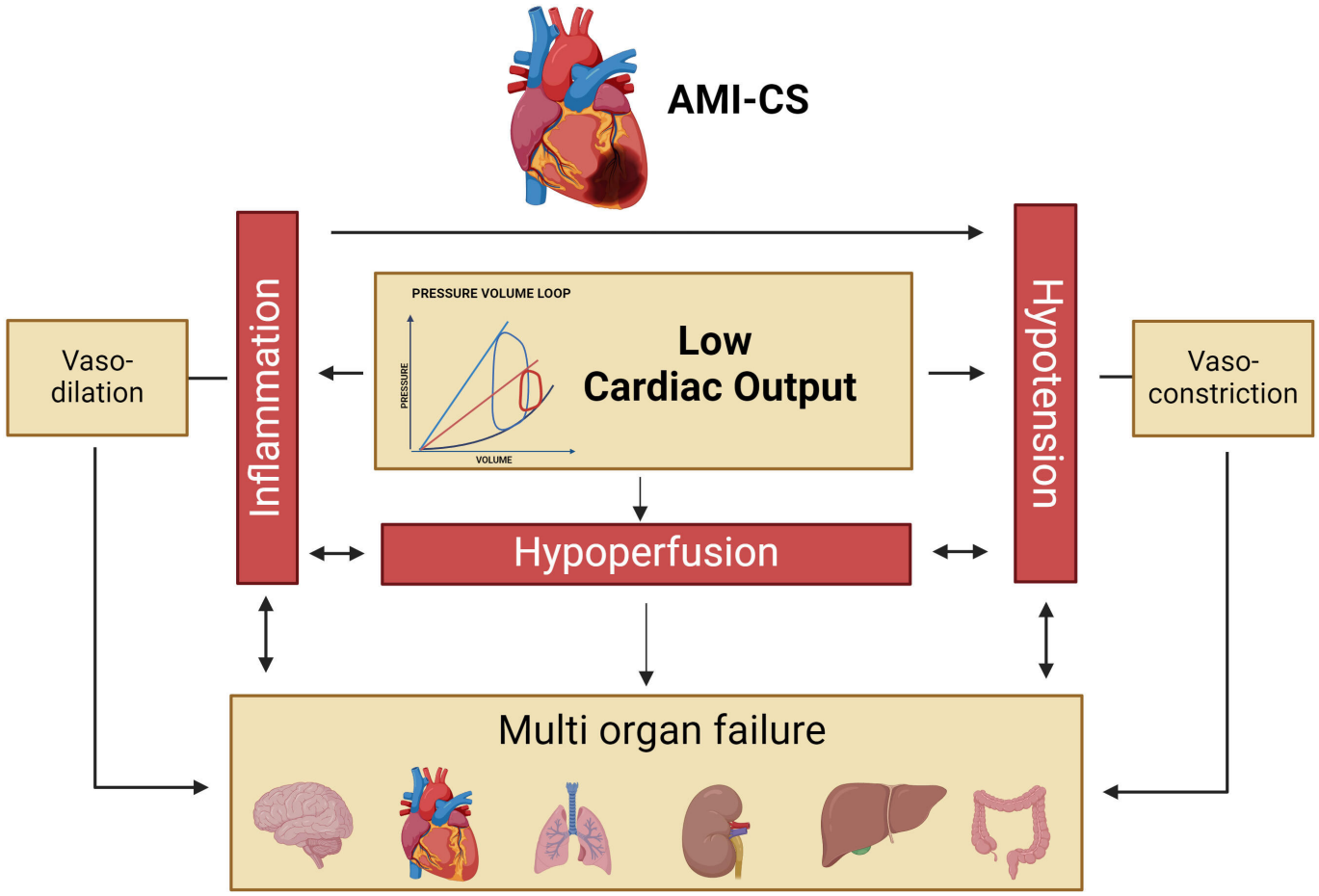

Figure 1 The pathophysiology of acute myocardial infarction-related cardiogenic shock (AMI-CS) begins with an abrupt loss of myocardial contractility, resulting in decreased cardiac output and systemic hypoperfusion, which will trigger a cascade of compensatory mechanisms. To maintain perfusion, endogenous catecholamines are released, initially increasing blood pressure, heart rate and vascular resistance. However, prolonged exposure to catecholamines depletes myocardial oxygen, worsening contractile dysfunction, organ hypoperfusion, and leading to metabolic acidosis. As a result, multiple organ systems—including the kidneys, liver and brain—are compromised. Inflammation and endothelial dysfunction further complicate the clinical course.

where it may offer most benefit. Further, scores were developed to predict overall survival rather than benefit from MCS. Finally, there is currently limited integration of echocardiography into existing risk stratification and patient selection for MCS. Validated point of care biomarkers, including imaging metrics, which reliably identify patients at risk of deterioration and ideally potential benefit from MCS are needed.

The role of pulmonary artery catheterisation (PAC) is also a source of debate. PAC is reasonable to guide device settings, identify potential need for escalation and evaluate signs of myocardial recovery, but its use is based on local practice and expert consensus. An ongoing trial in the USA (NCT05485376) is currently assessing the effect of PAC in an open-label randomised trial in cardiogenic shock.

HAEMODYNAMIC EFFECTS OF MCS DEVICES

MCS devices have fundamentally different profiles where some devices unload the heart by lowering ventricular filling pressure as opposed to devices that load the heart and increase filling pressure; the intra-aortic balloon pump (IABP) and microaxial flow pump (mAFP) are considered unloading devices opposed to venoarterial extracorporeal membrane oxygenation (V-A ECMO).

Intra-aortic balloon pump

In 1968, Adrian Kantrowitz reported the first successful use of counterpulsation in a patient with cardiogenic shock.[11] The IABP is a passive MCS device typically inserted via the femoral artery, but it can also be placed via the axillary artery. The 30–50 mL balloon inflates during diastole and deflates just before systole begins. During deflation, the balloon reduces aortic volume, thereby decreasing left ventricular afterload (table 1). The inflation of the balloon may thus increase diastolic pressure and enhance coronary and cerebral artery perfusion. However, in AMI-CS randomised data have failed to demonstrate any haemodynamic effect of IABP compared with inotropes alone.[12 13]

Microaxial flow pump

The left-sided mAFP devices, developed more than 25 years ago, are small catheter-based axial flow pumps inserted percutaneously through the femoral or surgically via the axillary artery and advanced retrogradely across the aortic valve into the LV.[14] Two main left-sided mAFP devices are currently available: Impella CP, which can be inserted percutaneously, and the Impella 5.5, which requires surgical placement via a graft. Once deployed, a fast-rotating impeller, driven by a miniature motor positioned on the catheter, will draw blood from the LV, expelling it into

Table 1 Main characteristics of most frequently used MCS systems in infarct-related cardiogenic shock

Device	IABP	V-A ECMO	Impella CP	ECMELLA
Main effect	Passive device with diastolic augmentation and systolic unloading	External centrifugal pump with retrograde aortic flow	Intracardiac axial flow pump unloading LV by aspiration of LV blood	Combination of V-A ECMO and intracardiac axial flow pump
Effect on cardiac output	↔	↑↑↑↑	↑↑↑	↑↑↑↑
Effect on LV filling pressure	↔	↑↑	↓↓↓	↓↓↓
Effect on RV filling pressure	↔	↓↓	↔	↓↓
LV myocardial oxygen consumption	↔	↑↔	↓↓	↓↓
Effect on systolic blood pressure	↓	↑↑	↔↓	↑
Oxygenation	No	Yes	No	Yes
Risk of Harlequin syndrome	No	Yes	No	Yes
Anticoagulation	No*	Yes	Yes	Yes
Duration of support	Days	Days	Days	Days
Arterial access size	8 Fr	15–21 Fr	14 Fr for placement, 9 Fr during support	2 arterial accesses both for Impella and ECMO cannula.
Antegrade limb perfusion	No	Standard	Can be done	Standard
Risk of lower limb ischaemia	(↑)	↑↑	↑	↑↑↑↑
Risk of bleeding complication	(↑)	↑↑↑	↑↑	↑↑↑↑
Risk of haemolysis	↔	↑	↑↑	↑
Risk of LV thrombus	↔	↑	↔	↔
Risk of acute kidney injury	↔	↔	↑	?
Device-specific contraindications	AR, PAD	AR, PAD	AR, PAD, mechanical aortic prosthesis, LV thrombus	AR, PAD, mechanical aortic prosthesis, LV thrombus

AR, moderate or severe aortic regurgitation; ECMELLA, combination of V-A ECMO and an Impella device; IABP, intra-aortic balloon pump; LV, left ventricular; MCS, mechanical circulatory support; PAD, peripheral arterial disease; RV, right ventricular; V-A ECMO, venoarterial extracorporeal membrane oxygenation.

the ascending aorta. The Impella CP may provide flow up to 4 L/min, while the Impella 5.5 may deliver more than 5 L/min. mAFPs reduce both the volume and pressure within the LV, decreasing left ventricular end-diastolic pressure (LVEDP), wall stress, and improving cardiac output.[15] This will lead to reduced workload and myocardial oxygen demand, which in experimental studies increased myocardial salvage following coronary occlusion.[16 17] Seyfarth and colleagues[18] demonstrated greater increases in cardiac index and more rapid lactate clearance with Impella 2.5 compared with IABP in 26 patients with AMI-CS. This was recently confirmed in Danish German Cardiogenic Shock (DanGer Shock) trial where the mAFP led to faster lactate clearance in STEMI-CS.[19]

Venoarterial extracorporeal membrane oxygenation

Extracorporeal life support in the V-A ECMO configuration provides combined cardiac and respiratory support, which is typically established percutaneously in AMI-CS via ultrasound-guided femoral vein and artery puncture. In adults, venous blood is drained through a large cannula (21–25 Fr) placed with the tip in the superior vena cava and returned via an arterial cannula (15–19 Fr) placed in the femoral artery. For femoral cannulation, an antegrade perfusion catheter is required to ensure adequate limb perfusion distal to the arterial cannula.[20] V-A ECMO unloads the right ventricle, but retrograde arterial flow increases left ventricular afterload, potentially leading to left ventricular distension and pulmonary congestion. In this situation, venting strategies, including IABP, transseptal puncture or mAFP, aim to decompress the LV.[21] In the Extracorporeal Life Support in Infarct-Related Cardiogenic Shock (ECLS-SHOCK) trial, active venting was used in only 6% of patients allocated to V-A ECMO.[22] V-A ECMO combined with mAFP (ECMELLA, table 1) has shown potential survival benefit in observational studies, but the configuration significantly raises the risk of complications and costs.[23] Two randomised trials on venting strategies using routine left atrial cannulation have been neutral.[24 25] Upcoming trials including UNLOAD-ECMO (NCT05577195) will offer further clarity. Until then, left ventricular venting should be reserved for selected cases with clear signs of left ventricular distension, rather than routine use.[26]

KEY CONSIDERATIONS FOR PATIENT AND DEVICE SELECTION

Choosing the appropriate MCS device depends on the patient's clinical status, medical history, age, haemodynamic profile and the presence of multiorgan failure. The use of a shock team in cardiogenic shock management ensures a multidisciplinary, coordinated approach to optimise rapid diagnosis and treatment, integrating expertise from cardiology, critical care and often cardiac surgery. This collaborative model has been shown to improve patient outcomes in observational studies and is recommended based on expert consensus.[27] Each device has specific contraindications for use (table 1). Given the heterogeneity of AMI-CS, accurately identifying the underlying pathophysiology is crucial for appropriate MCS selection where the primary goal is to restore systemic circulation and limit the doses of catecholamines and inotropes without damaging the heart and lungs when native cardiac output is inadequate. Therefore, confirming the presence of a low-flow state, characterised by low cardiac output alongside adequate or increased filling pressures, and evidence of hypoperfusion, is critical before initiating MCS (figure 2).

Initial assessment and echocardiography in AMI-CS

Assessment of hypoperfusion is traditionally based on clinical signs such as cold, mottled, clammy skin, oliguria, altered mental status and pulmonary congestion.[6 7 22 28] These may be non-specific and impractical in an emergency (oliguria). Arterial lactate will rise because of oxygen supply demand mismatch and is a strong predictor of outcome in patients with suspected

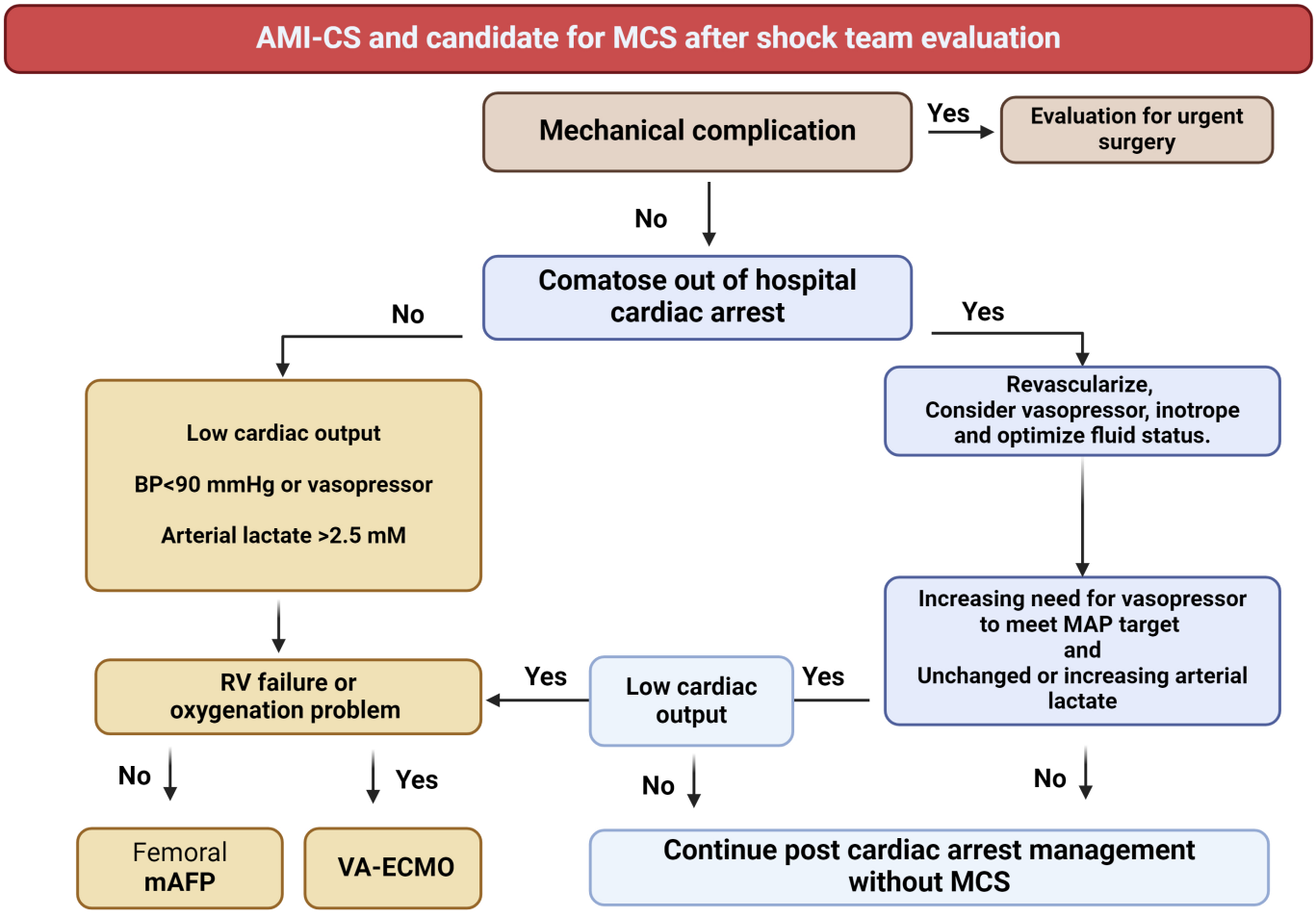

Figure 2 Suggested flow chart for selection of patients for mechanical circulatory support (MCS) with acute myocardial infarction-related cardiogenic shock (AMI-CS). Immediate assessment of cardiac output based on left ventricular (LV) outflow tract velocity-time integral, identification of contraindications for MCS placement and assessment of RV failure (generally a right ventricle exceeding the size of the left ventricle in apical four-chamber view, TAPSE <1 cm, and septal shift should raise concerns of severe RV failure). BP, blood pressure; mAFP, microaxial flow pump; MAP, mean reterial blood pressure; RV, right ventricular; TAPSE, tricuspid annular plane systolic excursion; V-A ECMO, venoarterial extracorporeal membrane oxygenation.

AMI when measured at arrival to catheterisation laboratory and should routinely be measured in unstable patients with AMI.[29]

Echocardiography plays a key role in the initial assessment of acute heart failure including AMI-CS.[30] It may support determination of shock aetiology, provide estimates of cardiac output and identify contraindications to MCS placement including left ventricular thrombus and severe aortic regurgitation, thus guiding MCS decisions.[31] While left ventricular ejection fraction is often used as a metric, it may be difficult to use this volumetric method for assessment of cardiac output in the emergency situation. A simple measure of stroke volume is the left ventricular outflow tract velocity-time integral (LVOT VTI), obtained via pulsed wave Doppler. A reduced LVOT VTI (typically <10–12 cm) suggests significantly decreased stroke volume.[26 31 32]

Direct measurement of LVEDP during coronary angiography will provide important information that can further refine the treatment. PAC, although valuable, may be too time consuming and should await revascularisation.

Evidence from clinical trials on MCS

Historically, IABP was the first and most widely used MCS device in AMI-CS. Its appeal lies in its relatively simple mechanism, which enhances coronary blood flow through diastolic augmentation and reduces afterload. This concept was first tested in the CRISP-AMI trial, where prophylactic use of IABP in patients with STEMI without shock prior to percutaneous coronary intervention (PCI) did not reduce infarct size when compared with immediate revascularisation (difference in infarct size 4.6% (95% CI −0.2% to 9.4%), p=0.06 in favour of immediate revascularisation).[33] In 600 patients with AMI-CS, the efficacy of IABP was tested in the IABP-SHOCK II trial (figure 3). The study included both STEMI and non-STEMI, allowed up to 30 min of resuscitation, and elevation of lactate was not mandatory.[28] Despite concerns regarding crossover rate (10% in the standard of care), enrolment of a heterogenous population with 45% having suffered OHCA and around 25% of patients having a lactate at randomisation <2.2 mmol/L, the study decisively demonstrated that IABP use did not reduce 30-day, 1-year or 6-year all-cause mortality and did not identify any subgroups that seemed to benefit from IABP.[28 34 35] The rate of bleeding complications and limb ischaemia was not increased with IABP. These findings led to a downgrade in the European guidelines, where IABP now carries a class III, level B recommendation for routine use in AMI-CS but retains a class 2a, level C recommendation in case of mechanical complications, based on expert consensus.[36] Despite the lack of benefit, IABP remains the most commonly used MCS device in the USA.[37]

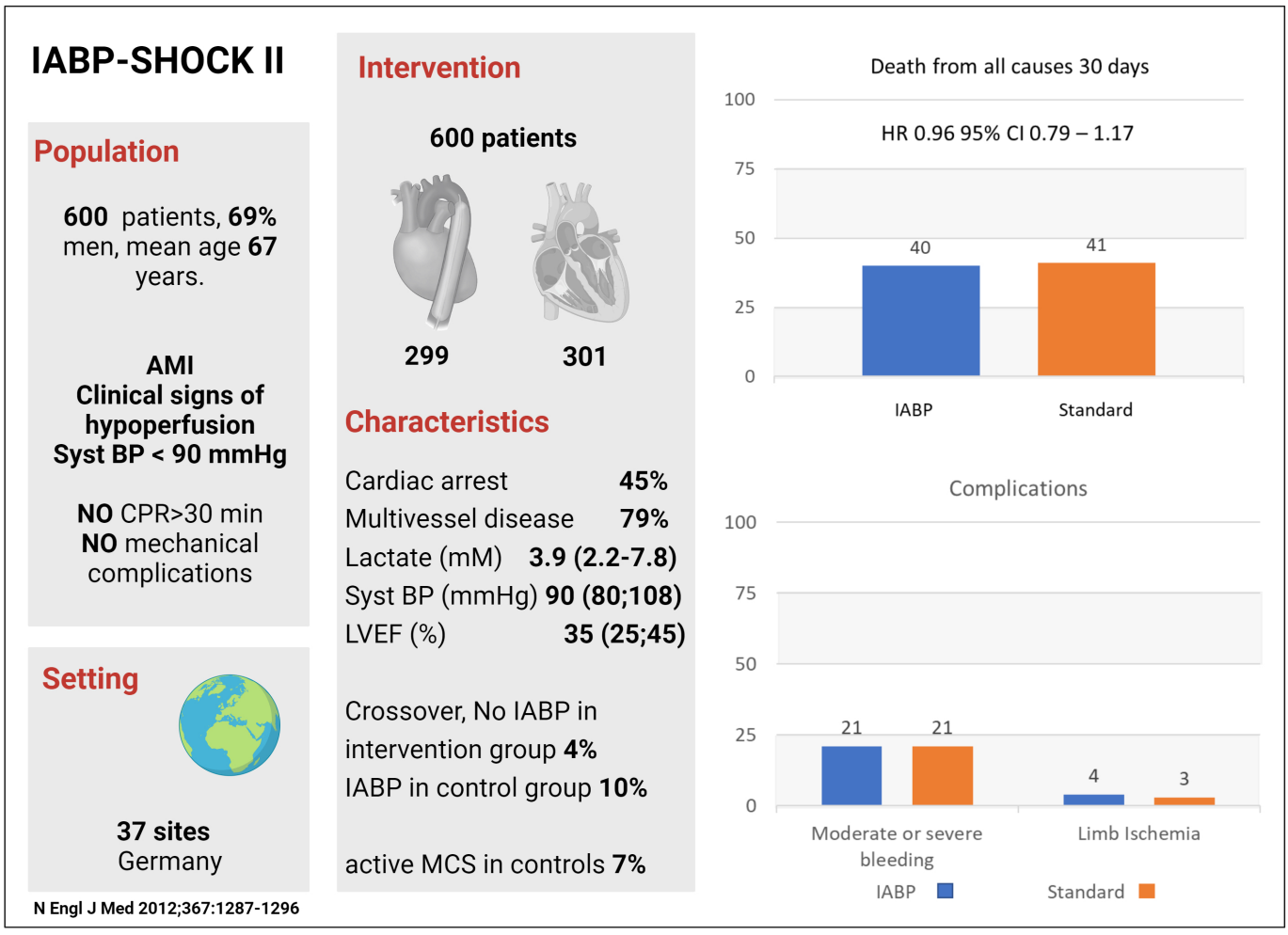

Figure 3 Summary of the Intra-aortic Balloon Support for Myocardial Infarction with Cardiogenic Shock (IABP-SHOCK II) trial. AMI, acute myocardial infarction; BP, blood pressure; CPR, cardiopulmonary resuscitation; IABP, intra-aortic balloon pump; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support.

During the last decade, the utilisation of V-A ECMO and mAFP has increased considerably. Use of V-A ECMO in AMI-CS has been investigated in several completed, early terminated and ongoing trials.[22 38 39] The largest randomised trial, the ECLS-SHOCK study, enrolled 417 patients to immediate V-A ECMO or standard of care in patients with AMI-CS planned for revascularisation with elevated lactate levels (>3 mmol/L), excluding resuscitation exceeding 45 min and mechanical infarct-related complications.[22] The trial found no significant difference in 30-day or 1-year mortality between V-A ECMO and standard of care, but found higher rates of limb ischaemia and bleeding complications with V-A ECMO (figure 4).[22 40] The study has mainly been criticised for 78% of patients having undergone resuscitation for cardiac arrest and low rate of left ventricular venting. A subsequent individual patient data meta-analysis of available trials confirmed no mortality benefit of V-A ECMO in AMI-CS.[7] These results have tempered enthusiasm for routine ECMO use in AMI-CS, although it remains an option in specific clinical scenarios (figure 2).

The mAFP has also been studied in AMI-CS. The IMPRESS in Severe Shock trial, which included 48 patients (95% of whom were postcardiac arrest), failed to show a mortality benefit of mAFP support.[41] The larger DanGer Shock trial enrolled 360 patients with STEMI-CS and predominantly left ventricular failure and a mandatory lactate elevation >2.5 mmol/L. Only patients with STEMI were enrolled and patients comatose after OHCA were excluded. The study started at four centres in Denmark and after 5 years the study was expanded to enrol patients in Germany and the UK completing after a total of 10 years of enrolment. The study demonstrated a statistically significant absolute 12.7% reduction in 180-day all-cause mortality with mAFP (figure 5). An exploratory post hoc analysis suggested this effect mainly was found in individuals younger than 77 years, whereas no effect of treatment was found in octogenarians or older.[42] Lactate clearance was faster in the mAFP group and requirement for vasoactive drugs less in the first hours of intensive care unit management.[19] The benefit came at the cost of increased rates of complications, including bleeding, limb ischaemia and sepsis. Further, risk of acute kidney injury and need for renal replacement therapy was increased in the mAFP group and linked to potentially modifiable factors such as suction events.[43] The study has been criticised for the long duration enrolment, difficult generalisability given the strict inclusion criteria and a high event rate in controls.[44]

The results of these landmark trials were recently merged in an individual patient data meta-analysis, combining data from 1059 patients randomised in nine active MCS versus control trials, with follow-up extended to 180 days.[6] Overall, MCS (of any type) did not reduce 6-month mortality (HR 0.87, 95% CI 0.74 to 1.03, p=0.10). However, targeting the STEMI-CS

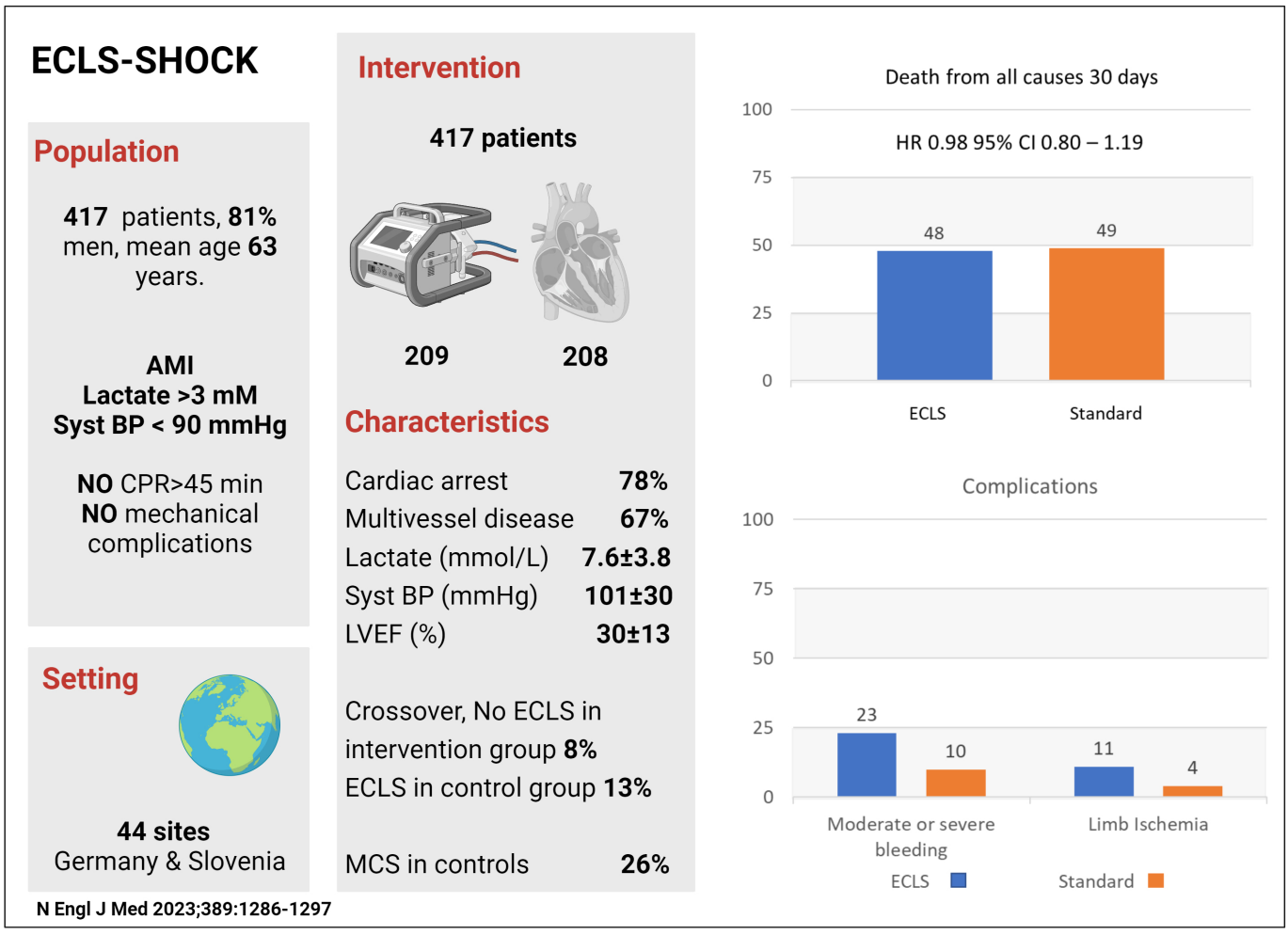

Figure 4 Summary of the Extracorporeal Life Support in Infarct-Related Cardiogenic Shock (ECLS-SHOCK) trial. AMI, acute myocardial infarction; BP, blood pressure; CPR, cardiopulmonary resuscitation; ECLS, extracorporeal life support; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support.

phenotype with a reduced risk of hypoxic brain injury defined as having suffered no cardiac arrest, having suffered cardiac arrest with regained consciousness or having cardiac arrest with a maximum duration of chest compression of 10 min demonstrated a mortality reduction with temporary MCS use (HR 0.77, 95% CI 0.61 to 0.97, p=0.024) regardless of the device type among 589 patients.

MCS timing

Optimal timing of MCS initiation remains controversial. Observational studies suggest that early MCS placement, particularly mAFP placement before revascularisation, may improve outcomes.[45] However, assessing this in an observational study introduces uncontrolled bias,[46] and none of the randomised trials to date were designed to specifically assess the impact of prerevascularisation versus postrevascularisation MCS. Until further randomised data are available, early MCS placement remains a consideration in patients with severe haemodynamic compromise, who are at high risk of further deterioration during reperfusion, and selected patients with refractory cardiac arrest.

MCS IN INFARCT-RELATED MECHANICAL COMPLICATIONS

Infarct-related mechanical complications, such as ventricular septal rupture and papillary muscle rupture, are life-threatening events that often necessitate urgent MCS to stabilise the patient before definitive surgical repair.[47] IABP is often used in these cases, and it retains a class 2a recommendation,[36] based on expert consensus. Early stabilisation with V-A ECMO, followed by delayed surgery, has been advocated by some experts. This approach allows time to optimise the patient's condition before undertaking surgical or percutaneous repair.[47] Due to the rarity of these conditions, large-scale randomised trials are unlikely to be conducted, leaving clinicians to rely on small uncontrolled studies, registry data and clinical experience.

COST-EFFECTIVENESS

The IABP is relatively inexpensive compared with other MCS devices. However, given the lack of demonstrable benefit in reducing mortality or reducing hospital length, it is not a cost-effective treatment in AMI-CS. The Impella is significantly more expensive than the IABP, with an estimated device cost of $20 000–$25 000 per device.[3 48] Even with a number needed to treat of 8 in the DanGer Shock, costs are substantial, and heath economic analyses are warranted. Even though the cost of oxygenator, tubes and cannulas for V-A ECMO is less than an mAFP, V-A ECMO is a resource-intensive and costly intervention, potentially exceeding the cost of mAFP treatment.[3 48]

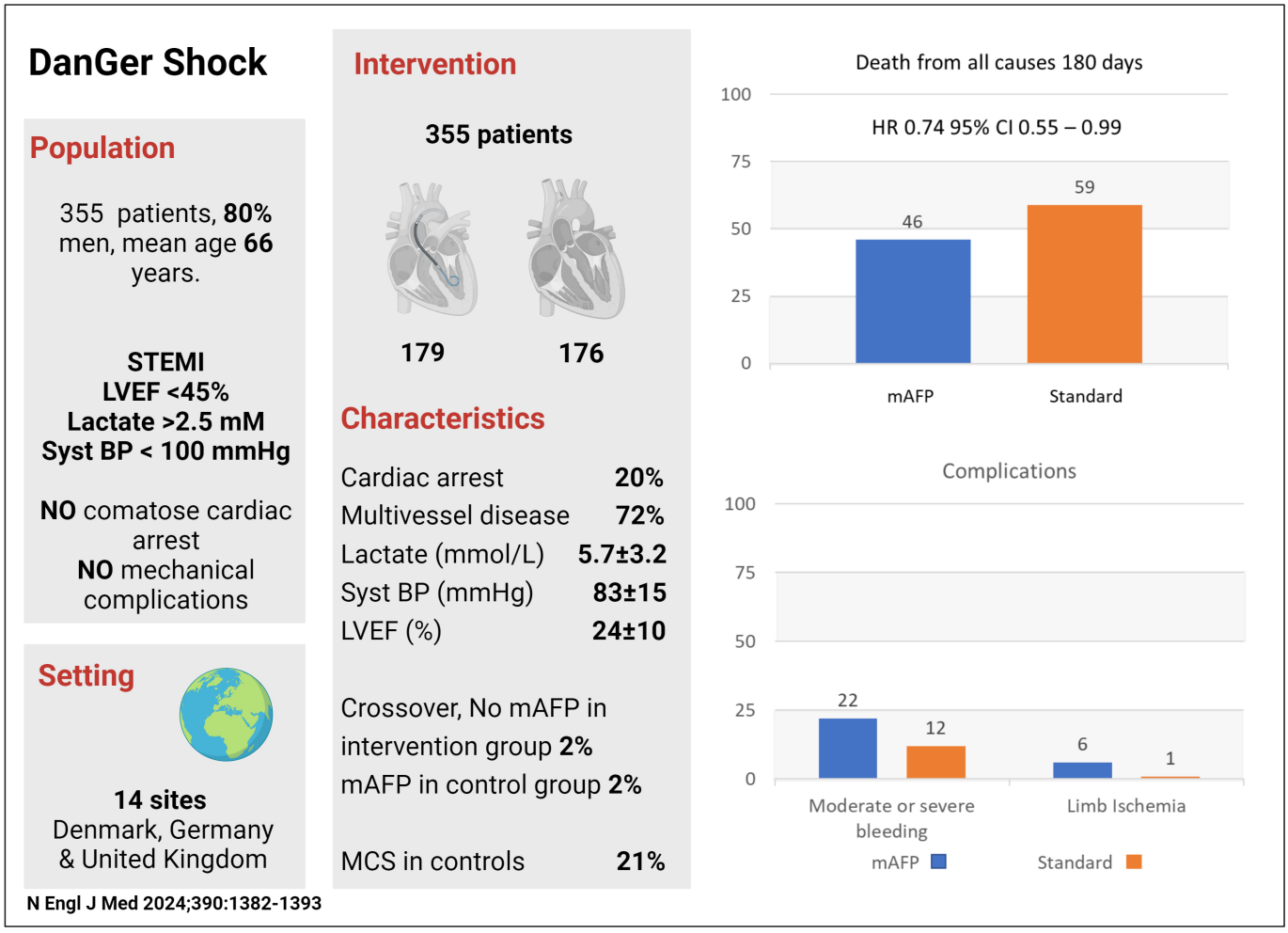

Figure 5 Summary of the Danish German Cardiogenic Shock (DanGer Shock) trial. BP, blood pressure; LVEF, left ventricular ejection fraction; mAFP, microaxial flow pump; MCS, mechanical circulatory support; STEMI, ST-segment elevation myocardial infarction.

FUTURE RESEARCH

Although some data suggest benefits of mAFP in selected patients with STEMI-CS, these results are somewhat fragile, relying on data from selected sites and healthcare systems that may be difficult to generalise. Despite a positive outcome in one study, further trials are necessary to confirm the potential benefits of MCS in AMI-CS and to address the potential benefit from V-A ECMO. The RECOVER IV trial (NCT05506449) sponsored by Abiomed/Johnson & Johnson planned to test haemodynamic support with an Impella-based treatment strategy, initiated pre-PCI in STEMI-CS. After publication of DanGer Shock, the data and safety monitoring board of RECOVER IV expressed concerns about equipoise, and the study was terminated. This has been questioned given the fragility of DanGer Shock and it would be welcomed to generate more data on benefit of routine use of MCS in selected patients with AMI-CS compared with a strategy with more conservative use. Two ongoing trials in AMI-CS will provide further insight. The ANCHOR trial (NCT04184635) targets to enrol 400 patients with AMI-CS allowing up to 30 min resuscitation and randomising to routine V-A ECMO or a non-MCS strategy excluding mAFP or other devices, but with the option of protocolised V-A ECMO rescue. The primary endpoint is death at day 30 in the V-A ECMO group and death or V-A ECMO rescue at day 30 in the control group. The ongoing ULYSS trial (NCT05366452) aims to enrol 204 patients with AMI-CS (elevation of lactate not mandatory), excluding patients with OHCA and resuscitation >10 min and test routine use of Impella CP versus standard of care.[49] The primary endpoint is a composite of all-cause mortality, use of ECMO rescue, left ventricular assist device or heart transplantation at 1 year. These studies will provide further important insights into the role of MCS in AMI-CS, but results are not anticipated before 2026–2027. Future studies should focus on refinement in patient selection and the avoidance of complications. Rapid bedside diagnostics to detect early signs of device-related complications, potentially driven by artificial intelligence algorithms, are an additional unmet need to maximise the benefit of these technologies.

Thus, diverse phenotypes and underlying causes complicate patient selection in clinical trials; ethical challenges also arise due to the severity of the condition, making timely informed consent and randomised controlled trial execution difficult. To overcome these hurdles, interdisciplinary collaboration across centres and countries is essential to achieve sufficient sample sizes. The development by industry partners of smaller, safer, easier to use and more advanced pumps, rigorously tested in randomised controlled trials rather than observational studies, is imperative.

Contributors JEM wrote the first draft of this review and all authors contributed to revision and acceptance of the final version.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests JEM received institutional research grants from Novo Nordisk Foundation and Abiomed; consulting fee from Boston Scientific; and speaker fees from Abbott, Abiomed and Boehringer Ingelheim. HT is the current president of the German Cardiac Society. UZ received institutional research grants from Pfizer, AstraZeneca and German Heart Foundation and speaker fees from Amgen, Bayer, BMS, Boehringer Ingelheim, Pfizer, Ferrer, Chiesi, Meril, Sanofi, Getinge, Fresenius and Zoll. AP received institutional funding from Getinge and Abiomed. CH received institutional research grants from the Novo Nordisk Foundation, Lundbeck Foundation and Danish Heart Foundation.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

Provenance and peer review Commissioned; externally peer reviewed.

ORCID iDs
Jacob Eifer Moller http://orcid.org/0000-0003-2873-5845
Holger Thiele http://orcid.org/0000-0001-7165-6478

REFERENCES

1 Helgestad OKL, Josiassen J, Hassager C, *et al*. Temporal trends in incidence and patient characteristics in cardiogenic shock following acute myocardial infarction from 2010 to 2017: a Danish cohort study. *Eur J Heart Fail* 2019;21:1370–8.
2 Jentzer JC, van Diepen S, Barsness GW, *et al*. Cardiogenic Shock Classification to Predict Mortality in the Cardiac Intensive Care Unit. *J Am Coll Cardiol* 2019;74:2117–28.
3 Bogerd M, Ten Berg S, Peters EJ, *et al*. Impella and venoarterial extracorporeal membrane oxygenation in cardiogenic shock complicating acute myocardial infarction. *Eur J Heart Fail* 2023;25:2021–31.
4 Josiassen J, Helgestad OKL, Møller JE, *et al*. Cardiogenic shock due to predominantly right ventricular failure complicating acute myocardial infarction. *Eur Heart J Acute Cardiovasc Care* 2021;10:33–9.
5 Link A, Pöss J, Rbah R, *et al*. Circulating angiopoietins and cardiovascular mortality in cardiogenic shock. *Eur Heart J* 2013;34:1651–62.
6 Thiele H, Møller JE, Henriques JPS, *et al*. Temporary mechanical circulatory support in infarct-related cardiogenic shock: an individual patient data meta-analysis of randomised trials with 6-month follow-up. *Lancet* 2024;404:1019–28.
7 Zeymer U, Freund A, Hochadel M, *et al*. Venoarterial extracorporeal membrane oxygenation in patients with infarct-related cardiogenic shock: an individual patient data meta-analysis of randomised trials. *Lancet* 2023;402:1338–46.
8 Josiassen J, Lerche Helgestad OK, Møller JE, *et al*. Hemodynamic and metabolic recovery in acute myocardial infarction-related cardiogenic shock is more rapid among patients presenting with out-of-hospital cardiac arrest. *PLoS One* 2020;15:e0244294.
9 Beske RP, Søndergaard FT, Møller JE, *et al*. Treatment effects of blood pressure targets and hemodynamics according to initial blood lactate levels in comatose out-of-hospital cardiac arrest patients - A sub study of the BOX trial. *Resuscitation* 2024;194.
10 Mieritz HB, Povlsen AL, Linde L, *et al*. DIFFERENCES IN MANAGEMENT AND PROGNOSTICATION OF CARDIOGENIC SHOCK PATIENTS IN THE PRESENCE AND ABSENCE OF OUT-OF-HOSPITAL CARDIAC ARREST. *Shock* 2024;61:209–14.
11 Yahr WZ, Butner AN, Krakauer JS, *et al*. Intraaortic phase-shift balloon pumping in the treatment of cardiogenic shock. *J Assoc Adv Med Instrum* 1969;3:100–5.
12 Prondzinsky R, Lemm H, Swyter M, *et al*. Intra-aortic balloon counterpulsation in patients with acute myocardial infarction complicated by cardiogenic shock: the prospective, randomized IABP SHOCK Trial for attenuation of multiorgan dysfunction syndrome. *Crit Care Med* 2010;38:152–60.
13 Prondzinsky R, Unverzagt S, Russ M, *et al*. Hemodynamic effects of intra-aortic balloon counterpulsation in patients with acute myocardial infarction complicated by cardiogenic shock: the prospective, randomized IABP shock trial. *Shock* 2012;37:378–84.
14 Siess T, Nix C, Menzler F. From a lab type to a product: a retrospective view on Impella's assist technology. *Artif Organs* 2001;25:414–21.
15 Møller-Helgestad OK, Hyldebrandt JA, Banke A, *et al*. Impella CP or VA-ECMO in profound cardiogenic shock: left ventricular unloading and organ perfusion in a large animal model. *EuroIntervention* 2019;14:e1585–92.
16 Meyns B, Stolinski J, Leunens V, *et al*. Left ventricular support by catheter-mounted axial flow pump reduces infarct size. *J Am Coll Cardiol* 2003;41:1087–95.
17 Swain L, Reyelt L, Bhave S, *et al*. Transvalvular Ventricular Unloading Before Reperfusion in Acute Myocardial Infarction. *J Am Coll Cardiol* 2020;76:684–99.
18 Seyfarth M, Sibbing D, Bauer I, *et al*. A randomized clinical trial to evaluate the safety and efficacy of a percutaneous left ventricular assist device versus intra-aortic balloon pumping for treatment of cardiogenic shock caused by myocardial infarction. *J Am Coll Cardiol* 2008;52:1584–8.
19 Udesen NLJ, Beske RP, Hassager C, *et al*. Microaxial Flow Pump Hemodynamic and Metabolic Effects in Infarct-Related Cardiogenic Shock: A Substudy of the DanGer Shock Randomized Clinical Trial. *JAMA Cardiol* 2024.:e244197.
20 Bonicolini E, Martucci G, Simons J, *et al*. Limb ischemia in peripheral veno-arterial extracorporeal membrane oxygenation: a narrative review of incidence, prevention, monitoring, and treatment. *Crit Care* 2019;23:266.
21 Lüsebrink E, Binzenhöfer L, Kellnar A, *et al*. Venting during venoarterial extracorporeal membrane oxygenation. *Clin Res Cardiol* 2023;112:464–505.
22 Thiele H, Zeymer U, Akin I, *et al*. Extracorporeal Life Support in Infarct-Related Cardiogenic Shock. *N Engl J Med* 2023;389:1286–97.
23 Schrage B, Becher PM, Bernhardt A, *et al*. Left Ventricular Unloading Is Associated With Lower Mortality in Patients With Cardiogenic Shock Treated With Venoarterial Extracorporeal Membrane Oxygenation: Results From an International, Multicenter Cohort Study. *Circulation* 2020;142:2095–106.
24 Park H, Yang JH, Ahn J-M, *et al*. Early left atrial venting versus conventional treatment for left ventricular decompression during venoarterial extracorporeal membrane oxygenation support: The EVOLVE-ECMO randomized clinical trial. *Eur J Heart Fail* 2023;25:2037–46.
25 Saku K, Kakino T, Arimura T, *et al*. Left Ventricular Mechanical Unloading by Total Support of Impella in Myocardial Infarction Reduces Infarct Size, Preserves Left Ventricular Function, and Prevents Subsequent Heart Failure in Dogs. *Circ Heart Fail* 2018;11:e004397.
26 Møller JE, Sionis A, Aissaoui N, *et al*. Step by step daily management of short-term mechanical circulatory support for cardiogenic shock in adults in the intensive cardiac care unit: a clinical consensus statement of the Association for Acute CardioVascular Care of the European Society of Cardiology SC, the European Society of Intensive Care Medicine, the European branch of the Extracorporeal Life Support Organization, and the European Association for Cardio-Thoracic Surgery. *Eur Heart J Acute Cardiovasc Care* 2023;12:475–85.
27 Chieffo A, Dudek D, Hassager C, *et al*. Joint EAPCI/ACVC expert consensus document on percutaneous ventricular assist devices. *Eur Heart J Acute Cardiovasc Care* 2021;10:570–83.
28 Thiele H, Zeymer U, Neumann F-J, *et al*. Intraaortic balloon support for myocardial infarction with cardiogenic shock. *N Engl J Med* 2012;367:1287–96.
29 Frydland M, Møller JE, Wiberg S, *et al*. Lactate is a Prognostic Factor in Patients Admitted With Suspected ST-Elevation Myocardial Infarction. *Shock* 2019;51:321–7.
30 McDonagh TA, Metra M, Adamo M, *et al*. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur Heart J* 2021;42:3599–726.
31 Tavazzi G, Price S, Beitnes JO, *et al*. Imaging in acute percutaneous mechanical circulatory support in adults: a clinical consensus statement of the Association for Acute CardioVascular Care (ACVC) of the ESC, the European Association of Cardiovascular Imaging (EACVI) of the ESC and the European branch of the Extracorporeal Life Support Organization (EuroELSO). *Eur Heart J Cardiovasc Imaging* 2024;25:e296–311.
32 Villavicencio C, Daniel X, Cartanyá M, *et al*. CARDIAC OUTPUT IN CRITICALLY ILL PATIENTS CAN BE ESTIMATED EASILY AND ACCURATELY USING THE MINUTE DISTANCE OBTAINED BY PULSED-WAVE DOPPLER. *Shock* 2023;60:553–9.
33 Patel MR, Smalling RW, Thiele H, *et al*. Intra-aortic balloon counterpulsation and infarct size in patients with acute anterior myocardial infarction without shock: the CRISP AMI randomized trial. *JAMA* 2011;306:1329–37.
34 Thiele H, Zeymer U, Neumann F-J, *et al*. Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. *Lancet* 2013;382:1638–45.
35 Thiele H, Zeymer U, Thelemann N, *et al*. Intraaortic Balloon Pump in Cardiogenic Shock Complicating Acute Myocardial Infarction: Long-Term 6-Year Outcome of the Randomized IABP-SHOCK II Trial. *Circulation* 2019;139:395–403.
36 Byrne RA, Rossello X, Coughlan JJ, *et al*. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J* 2023;44:3720–826.
37 Dhruva SS, Ross JS, Mortazavi BJ, *et al*. Use of Mechanical Circulatory Support Devices Among Patients With Acute Myocardial Infarction Complicated by Cardiogenic Shock. *JAMA Netw Open* 2021;4:e2037748.
38 Banning AS, Sabaté M, Orban M, *et al*. Venoarterial extracorporeal membrane oxygenation or standard care in patients with cardiogenic shock complicating acute myocardial infarction: the multicentre, randomised EURO SHOCK trial. *EuroIntervention* 2023;19:482–92.
39 Ostadal P, Rokyta R, Karasek J, *et al*. Extracorporeal Membrane Oxygenation in the Therapy of Cardiogenic Shock: Results of the ECMO-CS Randomized Clinical Trial. *Circulation* 2023;147:454–64.
40 Desch S, Zeymer U, Akin I, *et al*. Routine extracorporeal life support in infarct-related cardiogenic shock: 1-year results of the ECLS-SHOCK trial. *Eur Heart J* 2024;45:4200–3.
41 Ouweneel DM, Eriksen E, Sjauw KD, *et al*. Percutaneous Mechanical Circulatory Support Versus Intra-Aortic Balloon Pump in Cardiogenic Shock After Acute Myocardial Infarction. *J Am Coll Cardiol* 2017;69:278–87.
42 Klein A, Beske RP, Hassager C, *et al*. Treating Older Patients in Cardiogenic Shock with a Microaxial Flow Pump: Is it DANGERous? *J Am Coll Cardiol* 2024.
43 Zweck E, Hassager C, Beske RP, *et al*. Microaxial Flow Pump Use and Renal Outcomes in Infarct-Related Cardiogenic Shock - a Secondary Analysis of the DanGer Shock Trial. *Circulation* 2024;150:1990–2003.
44 Lüsebrink E, Binzenhöfer L, Thiele H. The DanGer Shock trial: a new dawn but much to uncover. *Eur Heart J* 2024;45:4181–3.

45 Iannaccone M, Franchin L, Hanson ID, *et al*. Timing of impella placement in PCI for acute myocardial infarction complicated by cardiogenic shock: An updated meta-analysis. *Int J Cardiol* 2022;362:47–54.
46 Almarzooq ZI, Song Y, Dahabreh IJ, *et al*. Comparative Effectiveness of Percutaneous Microaxial Left Ventricular Assist Device vs Intra-Aortic Balloon Pump or No Mechanical Circulatory Support in Patients With Cardiogenic Shock. *JAMA Cardiol* 2023;8:744–54.
47 Schlotter F, Huber K, Hassager C, *et al*. Ventricular septal defect complicating acute myocardial infarction: diagnosis and management. A Clinical Consensus Statement of the Association for Acute CardioVascular Care (ACVC) of the ESC, the European Association of Percutaneous Cardiovascular Interventions (EAPCI) of the ESC and the ESC Working Group on Cardiovascular Surgery. *Eur Heart J* 2024;45:2478–92.
48 Delmas C, Pernot M, Le Guyader A, *et al*. Budget Impact Analysis of Impella CP Utilization in the Management of Cardiogenic Shock in France: A Health Economic Analysis. *Adv Ther* 2022;39:1293–309.
49 Delmas C, Laine M, Schurtz G, *et al*. Rationale and design of the ULYSS trial: A randomized multicenter evaluation of the efficacy of early Impella CP implantation in acute coronary syndrome complicated by cardiogenic shock. *Am Heart J* 2023;265:203–12.